Exhibit 4

Specific Causation Expert Report for Jimmy Laramore
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The opinions of Dr. Reynolds, Dr. Hatten, and Dr. Bird confirm that Ms. Dyer's exposure to the chemicals at Camp Lejeune has been documented in other literature to have a positive association with the diagnosis of bladder cancer.

VI. General causation

Before advancing to the application of a differential etiology for Mr. Laramore, it is important to first recognize whether there is enough evidence to establish whether the chemicals in the water at Camp Lejeune are capable of causing bladder cancer as a general matter.

Numerous regulatory and scientific bodies have recognized that these four chemicals are toxic and capable of causing cancer. IARC recognizes TCE, vinyl chloride, and benzene as having sufficient evidence for carcinogenicity in humans, and that that PCE is probably carcinogenic to humans. IARC noted that the bladder "may be [a] target tissue[] for tetrachlorethylene-induced carcinogenesis in humans…"[49] EPA concluded that "TCE is carcinogenic to humans by all routes of exposure," that is, by ingestion, inhalation, and dermal exposure.[50] Further, EPA concluded that PCE is "likely to be carcinogenic in humans by all routes of exposure" by EPA.[51] Similarly, the National Toxicology Program has recognized TCE as "a known human carcinogen"[52] and PCE as "reasonably anticipated to be a human carcinogen."[53] ATSDR's 2017 Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases found sufficient evidence exists for PCE causing bladder cancer, stating that "the epidemiological studies provide sufficient evidence for causation and are consistent with the mechanistic information that certain genetic polymorphism may enhance the production of genotoxic PCE metabolites in the bladder via the GSH conjugate pathway." While ATSDR did not find sufficient evidence for TCE and bladder cancer, later studies have strengthened the association as noted by Dr. Hatten. As reported by Dr. Hatten and Dr. Plunkett, epidemiological studies have identified elevated bladder cancer diagnoses associated with benzene and vinyl chloride.[54]

As reported by Dr. Hatten, Dr. Plunkett, Dr. Gilbert, and Dr. Bird, both TCE and PCE share similar metabolic pathways: toxic metabolites are eventually excreted from

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[49] International Agency for Research on Cancer. Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014;106:1-514

[50] Environmental Protection Agency. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). 2011

[51] Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012

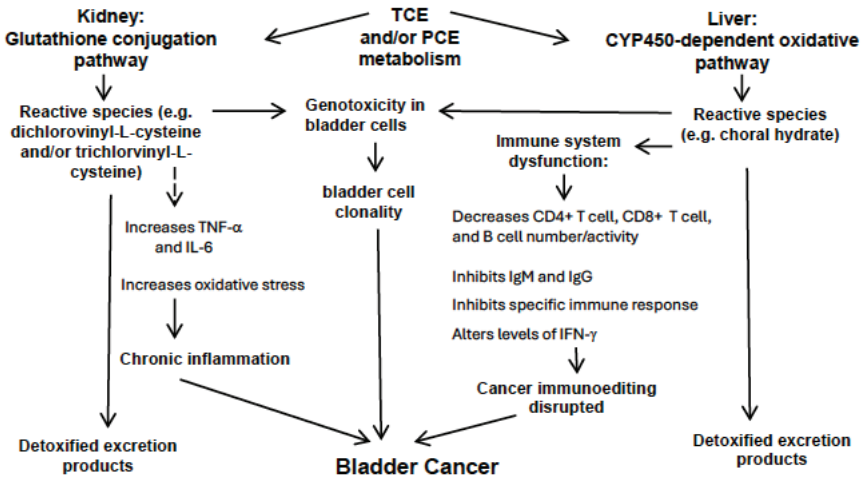
[52] National Toxicology Program (NTP). 2015. Report on Carcinogens monograph on trichloroethylene. Research Triangle Park, NC: National Toxicology Program. RoC Monograph 05

[53] NTP (National Toxicology Program). 2021. Report on Carcinogens, Fifteenth Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service

[54] Hatten pp. 26-29; 31-32; Plunkett ¶ 47

the kidneys into urine where it sits in the bladder until voided.[55] Dr. Plunkett identifies the same endpoint for benzene and vinyl chloride metabolites as well.[56] This means that the toxic metabolites can spend hours in contact with urothelial cells inside the bladder. Below is a figure from Dr. Gilbert explaining the metabolic pathways and outcome for TCE and PCE-induced bladder cancer

Figure 1. Model for TCE and/or PCE-induced bladder cancer



Dr. Gilbert reports that inhalation and dermal exposure from TCE-contaminated water at least doubles ingestion consumption figures (and with similar evidence for PCE).[57] Dr. Gilbert further explains that a mixture of TCE, PCE, and benzene can produce additive effects that can cause bladder cancer in that both TCE and PCE share a similar metabolic pathway and all three chemicals promote chronic inflammation and immunosuppression.[58] Regarding chronic inflammation in particular, Dr. Gilbert concludes that it "is an important driver of bladder cancer and provides support for tumor progression, metastasis, and anti-cancer resistance." In addition, TCE and PCE's can reduce the impact of the body's natural immune response to bladder cancer, which is important given that the most common intravesical treatment used to fight bladder cancer – BCG – essentially activates an adaptive immune response.[59]

Over time, the scientific consensus has progressed to greater certainty, and action, regarding the toxicity of the chemicals at Camp Lejeune. In December 2024 EPA finalized a rule banning on TCE and most commercial uses of PCE under the Toxic Substances Control Act, describing TCE as "extremely toxic" and PCE as "cancer-causing". As noted

[55] Plunkett ¶¶ 33, 43; Hatten p. 39; Bird pp. 17-18
[56] Plunkett ¶¶ 52, 56, 59
[57] Gilbert p. 30
[58] Gilbert p. 32-3
[59] Gilbert Rep. at p. 19-20

by Dr. Bird in his supplemental report, "the EPA determined that any *lesser* restrictions on the use of TCE or PCE would fail to adequately protect public health."[60] Dr. Bird further explained that EPA's safety measures were based on the wastewater concentrations, not consumption, meaning that the risk for those at Camp Lejeune (whose ingested concentrations alone are than the concentrations identified in the EPA rule) is even greater.

Accordingly, there is a sufficient basis to conclude that the chemicals in the water at Camp Lejeune are capable of causing bladder cancer.

VII. Differential etiology

The next step in this analysis is to perform a differential etiology on Mr. Laramore to determine if his exposure to the water at Camp Lejeune is at least as likely as not to cause his bladder cancer.

First, it is helpful to a general background on bladder cancer itself. Bladder cancer arises from the cells lining the urinary system including the bladder, ureter, renal pelvis and prostatic urethra, most commonly in the transitional epithelium (also known as urothelium). Bladder cancer is a disease of carcinogen exposure. As our body encounters toxins it must have mechanisms of which to remove them from the body. Once such mechanism is to filter them in our kidneys. In doing so the carcinogen/toxin is placed into urine for us to then void out of our system. The major issue with this process is that our bladder is a storage organ leading to the urine and carcinogen being exposed to the urothelial (lining of the bladder) in many cases for hours. The carcinogen contact/exposure to the urothelial then leads to the cellular damage, which eventually can lead to cancer formation. Bladder cancer can present as non-invasive bladder cancer (NMIBC) or muscle invasive bladder cancer (MIBC) approximately 70% and 25%, respectively or de novo metastatic cancer in 5%. The primary risk factors for bladder cancer include smoking, exposure to environmental and occupational carcinogens, age, gender, and certain medical conditions. While bladder cancer is treatable when detected early, late-stage diagnosis significantly reduces survival rates.

Bladder cancer can arise from the presence of a single risk factor or a combination of risk factors. Bladder cancer risk factors often interact in a way that magnifies an individual's overall risk, a phenomenon known as synergistic or cumulative risk. For instance, smoking is the most prominent risk factor for bladder cancer, with smokers being three to four times more likely to develop bladder cancer compared to non-smokers. However, when combined with environmental carcinogen exposure, such as that from industrial chemicals like benzene, trichloroethylene (TCE), or perchloroethylene (PCE), commonly found in workplaces or contaminated water supplies, the risk can be significantly higher. Studies show that individuals exposed to both smoking and toxic chemicals, such as those at Camp Lejeune, experience a greater risk of bladder cancer than the sum of the risks posed by each factor alone The interaction between these factors may increase the concentration of carcinogens in the

[60] Bird Suppl. p. 1